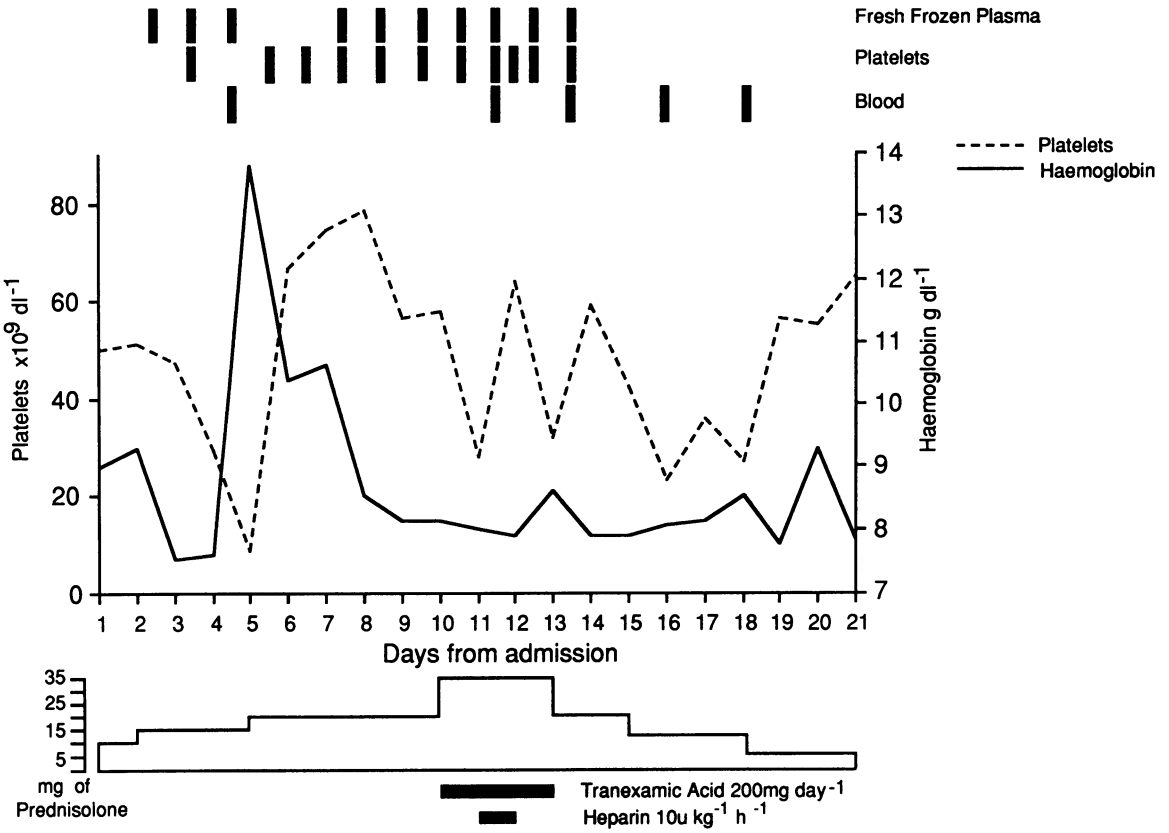

Figure 2. Chart showing the treatment of the severe bleeding episode and the response of the haemoglobin and platelet count

platelets 20×10/9 per litre and elevated fibrinogen degradation products 0.4 mg/dl. Occasional further episodes of minor subcutaneous haemorrhage have not necessitated admission. While learning to walk her prominent bruising caused her parents to find themselves the victims of misplaced speculation about child abuse, a problem helped by the provision of protective headgear.

Discussion

Kasabach-Merritt syndrome is rare. There is difficulty in distinguishing treatment response from spontaneous improvement. Platelet and clotting factor replacement is logical treatment in a bleeding patient with consumption coagulopathy and has been used with satisfactory results in many case reports. However, our patient had persistent life threatening subcutaneous bleeding which continued during 13 days of intensive platelet and clotting factor support. When this treatment was stopped due to lack of therapeutic effect clinical improvement was observed within 48 h.

This is the first report suggesting that platelet replacement may exacerbate Kasabach-Merritt syndrome. We believe that a closely monitored trial withdrawal of treatment is indicated in patients with Kasabach-Merritt who continue to bleed in spite of maximal therapy.

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'Ecstasy' induced pneumomediastinum

A J Levine BSc MBBS FRCS **S Drew** MBBS
G M Rees MS FRCS FRCP *Department of Cardiothoracic Surgery, St Bartholomew's Hospital, West Smithfield, London EC1*

Keywords: Ecstasy; Boerrhave's syndrome; pneumomediastinum; narcotic abuse

Case presented to Clinical Section, 12 June 1992

A 17-year-old man presented at an Accident and Emergency Department complaining of chest pain and vomiting. He gave a history of having ingested two tablets of 'Ecstasy' (an illegal narcotic, Methylenedioxymethamphetamine (MDMA)) 24 h earlier at a party and had begun vomiting some 2 h after ingestion. This vomiting persisted and about 12 h later he had experienced sudden retrosternal chest pain which had persisted (and increased in intensity) and odontophagia. He had no past medical history of note. On examination he was flushed, febrile (temperature 37.9) and tachycardic. He was noted to have surgical emphysema extending over his chest, neck and face. Examination of his cardiovascular system showed him to have a pericardial 'crunch' (Hamman's sign), examination of his respiratory system and abdomen was unremarkable. A clinical diagnosis of ruptured oesophagus (Boerrhave's syndrome) was made and the patient was transferred to a cardiothoracic unit.

Investigations on admission showed him to have a raised white blood cell count (19400) and gross pneumomediastinum and pneumoretroperitoneum. He was treated conservatively with analgesia, IV fluids, antibiotics (a cephalosporine and metronidazole) and put nil by mouth. An urgent gastrografin swallow and meal demonstrated no oesophageal leak.

Conservative management was continued and over the course of the next few days his symptoms and signs regressed. A repeat contrast study again demonstrated no leak and with resolution of his clinical picture his CXR improved. The patient was discharged 7 days after admission painfree and eating normally. Since this episode the patient has been asymptomatic and has taken no more MDMA.

Discussion

Pneumomediastinum is a relatively rare condition and when unassociated with obvious perforation of an intrathoracic viscus is termed 'spontaneous'. Spontaneous pneumomediastinum has been described in relation to many different circumstances including asthma, barotrauma secondary to artificial ventilation, laparoscopy[1], oesophagoscopy (without oesophageal perforation), transbronchial biopsy[2], dental extraction, sinus wash out[3], belching[4], violent exercise and drug abuse.

Pneumomediastinum in relation to drug abuse was first noted as a consequence of cocaine inhalation[5] and recently it has been observed in subjects inhaling alkaloid opiates and marijuana. In all these cases it is postulated that the mechanism of pneumomediastinum is an increase in intrathoracic pressure caused by the valsalva manoeuvre (performed by the subject to heighten the effect of the narcotic) leading to rupture of marginal alveoli and resultant 'back-tracking' of the air into the mediastinum via the perivascular spaces around the pulmonary artery. We believe this to be the first case described of an ingested narcotic causing pneumomediastinum and feel that the aetiology here is due to vomiting against a fixed glottis causing a rise in intrathoracic pressure but no oesophageal perforation.

'Ecstasy' use is associated with a number of clinico-pathological states, eg acute psychotic reactions, hyperthermia, hypotension, tachycardia, disseminated intravascular coagulation, adult respiratory distress syndrome (ARDS) and rhabdomyolosis leading to acute renal failure[6]. With its widespread use amongst many young people more sequelae will become manifest with time. We believe that faced with a young patient giving a history of chest (or neck) pain associated with narcotic abuse a physician should immediately get a chest X-ray, and if a pneumothorax, pneumomediastinum or pneumopericardium is noted an immediate contrast swallow should be arranged. If this demonstrates no oesophageal rupture a conservative policy should be adopted (as above) with gradual reintroduction of fluids and food by mouth as the patient clinically improves and the X-ray normalizes.

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'Ecstasy' ingestion: a case report of severe complications

P J Barrett MB ChB FCAnaes
G T Taylor MRCP(UK) FFARCS
Department of Anaesthesia, Broadgreen Hospital, Thomas Drive, Liverpool L14 3LB

Keywords: drug abuse; ecstasy; acute renal failure; disseminated intravascular coagulation; rhabdomyolysis

Ecstasy or MDMA (3,4,methylenedioxymethamphetamine) is a Class A controlled drug in the United Kingdom. This case report details the severe complications of disseminated intravascular coagulation (DIC), acute renal failure (ARF), rhabdomyolysis and impaired liver function following the ingestion of a single tablet of ecstasy and a quantity of amphetamine.

Case report

A 23-year-old male was admitted to casualty following a convulsion 3 h after the ingestion of one ecstasy tablet and £5 worth of amphetamine. Significant findings were: agitation, mydriasis, purposeless clonic movements, tachypnoea of 40/min, tachycardia of 130/min, blood pressure 120/80 mmHg, and axillary temperature 40°C. Immediate therapy included oxygen and chlorpromazine 50 mg intramuscularly on two occasions to control agitation. The patient subsequently developed a tachycardia of 160/min and his blood pressure fell to 85/60 mmHg, together with a reduced level of consciousness. Intravenous fluids rapidly restored his blood pressure to 100 mmHg systolic and propranolol 5 mg was administered incrementally to control tachycardia. He was intubated and transferred to intensive care.

On admission, he was sedated, ventilated and monitored invasively. Over 12 h, a progressive metabolic acidosis was noted and the naso-gastric aspirate was bloodstained. He developed haematuria and oliguria despite adequate fluid loading and blood pressure. Frusemide 40 mg was administered intravenously, initially with good effect, but there was biochemical and haematological evidence of rhabdomyolysis, renal and hepatic dysfunction and DIC (Table 1). Toxicology studies revealed an MDMA level of 0.2 mg/l and an amphetamine level of 0.1 mg/l. The patient was treated aggressively with blood and blood products and daily haemofiltration. He was extubated after three days, biochemical and haematological profiles improved, and haemofiltration was continued for 21 days, after which his renal function returned to normal. The patient was discharged from hospital 33 days after his admission.

Discussion

MDMA is a semi-synthetic hallucinogenic compound related to amphetamine and mescaline. In the UK, MDMA became established within drug using circles at so called 'Acid House' parties in the late 1980s. Little is known of the pharmacology and toxicology of MDMA, but the initial side effects are sympathomimetic[1]. A review of the literature revealed two fatalities in the UK associated with hyperthermia, rhabdomyolysis and coagulopathy[2,3] and four fatalities in the USA due to cardiac complications or accidents[4,5].

Our patients toxicology studies revealed non toxic levels of both MDMA and amphetamine, but the combined level

Correspondence to: Dr P J Barrett, 28 Rockingham Close, Gorse Covert, Warrington WA3 6UY